

## IN THE CLAIMS

### **Amendments to the Claims:**

Please amend claim 19 as indicated, below.

Please add new claims 46-59.

The following listing of claims will replace all prior versions and listings of claims in the application.

Following amendments, claims 19-30 and 46-59 will be pending in the application.

### **Listing of Claims:**

1-18 (Canceled).

19. (Currently amended) A method for treatment and/or prophylaxis of inflammation in a mammalian patient which method comprises administering to said patient an effective amount of apoptotic bodies, wherein said apoptotic bodies exhibit at least two characteristics comprising DNA fragmentation, surface exposure of phosphatidylserine, or altered mitochondrial membrane permeability, to up-regulate the *in vivo* generation of anti-inflammatory Th-2 derived cytokines and/or down regulate the *in vivo* generation of pro-inflammatory Th-1 derived cytokines thereby reducing the level of inflammation in the treated patient.

20. (Previously presented) The method of claim 19, wherein the apoptotic bodies are in a liquid suspension along with viable cells.

21. (Previously presented) The method of claim 20, wherein the apoptotic bodies comprise from 10% to 90% of the cellular portion of the suspension.

22. (Previously presented) The method of claim 21, wherein the apoptotic bodies comprise from 30% to 70% of the cellular portion of the suspension.

23. (Previously presented) The method of claim 19, wherein the apoptotic bodies are derived from extracorporeal treatment of blood cells compatible with those of the mammalian patient.

24. (Previously presented) The method of claim 19, wherein the apoptotic bodies are derived from established cultured cell lines.

25. (Previously presented) The method of claim 23, wherein the blood cells are white blood cells of blood compatible with that of the mammalian patient.

26. (Previously presented) The method of claim 25, wherein the blood cells are the patient's own white blood cells.

27. (Previously presented) The method of claim 26, wherein the blood cells are the patient's own T lymphocytes.

28. (Previously presented) The method of claim 19, further comprising administering to a human patient a dosage of apoptotic bodies comprising from 10,000 to 10,000,000 apoptotic bodies per kilogram body weight of the patient.

29. (Previously presented) The method of claim 28, wherein the dosage contains from 500,000 to 5,000,000 apoptotic bodies per kilogram body weight of the patient.

30. (Previously presented) The method of claim 28, wherein the dosage contains from 1,500,000 to 4,000,000 apoptotic bodies per kilogram body weight of the patient.

31-45 (Canceled).

46. (New) A method for treatment and/or prophylaxis of inflammation in a mammalian patient with an inflammatory disorder, wherein the disorder is selected from the group consisting of contact hypersensitivity, psoriasis, rheumatoid arthritis, scleroderma, lupus, diabetes mellitus, organ rejection, miscarriage, multiple sclerosis, inflammatory bowel disease, and graft versus host disease, which method comprises administering to said mammalian patient an effective amount of apoptotic bodies to up-regulate the *in vivo* generation of anti-inflammatory Th-2 derived cytokines and/or down regulate the *in vivo* generation of pro-inflammatory Th-1 derived cytokines thereby reducing the level of inflammation in the treated patient, and wherein said apoptotic bodies exhibit least two characteristics comprising DNA fragmentation, surface exposure of phosphatidylserine, or altered mitochondrial membrane permeability.

47. (New) The method of claim 46, wherein the apoptotic bodies are in a liquid suspension along with viable cells.

48. (New) The method of claim 47, wherein the apoptotic bodies comprise from 10% to 90% of the cellular portion of the suspension.

49. (New) The method of claim 48, wherein the apoptotic bodies comprise from 30% to 70% of the cellular portion of the suspension.

50. (New) The method of claim 46, wherein the apoptotic bodies are derived from extracorporeal treatment of blood cells compatible with those of the mammalian patient.

51. (New) The method of claim 46, wherein the apoptotic bodies are derived from established cultured cell lines.

52. (New) The method of claim 50, wherein the blood cells are white blood cells of blood compatible with that of the mammalian patient.

53. (New) The method of claim 52, wherein the blood cells are the patient's own white blood cells.

54. (New) The method of claim 53, wherein the blood cells are the patient's own T lymphocytes.

55. (New) The method of claim 46, further comprising administering to a human patient a dosage of apoptotic bodies comprising from 10,000 to 10,000,000 apoptotic bodies per kilogram body weight of the patient.

56. (New) The method of claim 55, wherein the dosage contains from 500,000 to 5,000,000 apoptotic bodies per kilogram body weight of the patient.

57. (New) The method of claim 55, wherein the dosage contains from 1,500,000 to 4,000,000 apoptotic bodies per kilogram body weight of the patient.

58. (New) A method for treatment and/or prophylaxis of inflammation in a mammalian patient which method comprises administering to said patient an effective amount of apoptotic bodies, wherein said apoptotic bodies exhibit at least two characteristics comprising the binding of Fas ligands to Fas receptors, caspase activation, DNA fragmentation, surface exposure of

phosphatidylserine, altered mitochondrial membrane permeability, or release of mitochondrial cytochrome-c, with consequent alleviation of patient's symptoms of congestive heart failure, to up-regulate the *in vivo* generation of anti-inflammatory Th-2 derived cytokines and/or down regulate the *in vivo* generation of pro-inflammatory Th-1 derived cytokines thereby reducing the level of inflammation in the treated patient.

59. (New) A method for treatment and/or prophylaxis of inflammation in a mammalian patient with an inflammatory disorder, wherein the disorder is selected from the group consisting of contact hypersensitivity, psoriasis, rheumatoid arthritis, scleroderma, lupus, diabetes mellitus, organ rejection, miscarriage, multiple sclerosis, inflammatory bowel disease, and graft versus host disease, which method comprises administering to said mammalian patient an effective amount of apoptotic bodies to up-regulate the *in vivo* generation of anti-inflammatory Th-2 derived cytokines and/or down regulate the *in vivo* generation of pro-inflammatory Th-1 derived cytokines thereby reducing the level of inflammation in the treated patient, wherein said apoptotic bodies exhibit at least two characteristics comprising the binding of Fas ligands to Fas receptors, caspase activation, DNA fragmentation, surface exposure of phosphatidylserine, altered mitochondrial membrane permeability, or release of mitochondrial cytochrome-c, with consequent alleviation of patient's symptoms of congestive heart failure,